**PERIPHERAL TRIGEMINAL STIMULATION-INDUCED MICROGLIAL ACTIVATION IN THE SECOND-ORDER NEURONS IN THE RAT**

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Migraine is a common neurovascular disorder characterized by paroxysmal headache attacks. The exact pathomechanism is still lacking, but the leading hypothesis targets the activation of the trigeminovascular system (TS). Recently the involvement of microglial cells has been emphasized in the central nociceptive processes. Microglial activation was detected in the dorsal horn of the spinal cord following the peripheral sensory nerve injury. Based on these findings, our goal is to study the role of glia in the TS activation related pain model.

Subcutaneous formalin solution (50 µl, 1.5%) was injected into the right whisker pad of young adult Sprague-Dawley rats. 4, 24 and 72 hours after injections, the animals were transcardially perfused and the caudal trigeminal nucleus (TNC) was removed. 30 µm slices were made by cryostat and the tissues were subjected to CD11b fluorescent immunohistochemistry.

We found uniformly distributed resting microglial cells (small cell body; long, thin multiple branches; fine ramifications) in the TNC of control (intact) animals, whereas activated microglial cells (hypertrophied, amoeboid form) were identified on the stimulated side of the TNC in the 72-hour treated animals. Significant quantitative changes and morphological transformations of the CD11b-immunopositive cells were not seen in the 4 and 24-hour treated rats compared to the control.

Our results are in keeping with changes observed in neuropathic pain models. Present work provides an opportunity to study the neuron-glia interactions in response to peripheral trigeminal triggers and allows testing new specific markers and metabolites of the kynurenine pathway.